Important Advances in Clinical Medicine

Epitomes of Progress—Anesthesiology

The Scientific Board of the California Medical Association presents the following inventory of items of progress in anesthesiology. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist the busy practitioner, student, research worker or scholar to stay abreast of these items of progress in anesthesiology which have recently achieved a substantial degree of authoritative acceptance, whether in his own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Anesthesiology of the California Medical Association and the summaries were prepared under its direction.

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Serum Potassium and Anesthesia

THE SERUM POTASSIUM concentration of surgical patients is of critical concern to anesthesiologists because of its effect on myocardial function. Potassium ions are the most important cations for maintaining resting membrane potential and repolarization of the cardiac cell. Furthermore, myocardial contractility is influenced by intracellular potassium concentration.

Potassium ions are 200 times more permeable (that is, mobile) than sodium across the cell membrane. This means that serum potassium concentrations are easily influenced by disease states, preoperative drug therapy, anesthetic management and the acid-base status of a patient. Potassium is the predominant intracellular cation while sodium is the predominant extracellular cation. The resting membrane potential is maintained by the transmembrane potassium concentration gradient of about 155 mEq per liter inside and 4 mEq per liter outside.

Clinically, only the serum potassium is easily measured, which may give rise to erroneous interpretation and mismanagement of the patient since it may not reflect the total potassium content of the body. Serum alkalemia is the most common cause of relative hypokalemia. When serum alkalemia is produced by hyperventilation (respiratory alkalosis) or by increased serum bicarbonate (metabolic alkalosis), the body attempts to compensate by increasing the number of hydrogen ions in the extracellular space and increasing renal excretion of bicarbonate ions. The former is a more immediate physiologic compensatory mechanism. The source of hydrogen ions recruited is from the intracellular compartment at the expense of an exchange with potassium ions from the extracellular compartment. Therefore, serum alkalemia can lead to hypokalemia without immediate loss of potassium from the body. Serum acidemia tends to produce a relative hyperkalemia through a reversal of the potassium-hydrogen ion exchange. Acute reduction of serum potassium, without loss of body potassium, is not detrimental to the cardiovascular system. However, in patients whose total body potassium has been depleted by diuretics, digitalis, gastrointestinal loss or endocrine disorders, sudden reduction in serum potassium from hyperventilation can produce serious cardiac dysrhythmias. Furthermore, most of the drugs (including anesthetics) administered by an anesthesiologist are cardiovascular depressants and they can potentiate the

already reduced cardiac output from cardiac dysrhythmias.

Acute increases in serum potassium can lead to cardiac conduction block and arrest. Acute hyperkalemia may be caused by inappropriate exogenous administration of potassium chloride or the use of succinylcholine for muscle relaxation in patients who are severely burned, traumatized or have spinal cord injuries. When the serum potassium is allowed to accumulate in the body from lack of elimination (that is, renal failure), the potassium ions tend to distribute in body compartments maintaining the normal transmembrane potassium concentration gradient. Therefore, in chronically hyperkalemic patients elevated serum potassium levels tend to be tolerated far better than in normal patients whose serum potassium concentration is subjected to an acute rise. Nevertheless, a serum potassium value greater than 10 mEq per liter usually leads to cardiac arrest regardless of whether the hyperkalemia is acute or chronic.

In summary, both hypokalemia and hyperkalemia can disturb electrophysiology and induce cardiac failure. It is important to accurately determine the cause of the serum potassium abnormality in order to institute appropriate management.

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Local Anesthetic Agents in Obstetrics—An Update

THE USE OF local anesthetic agents to relieve the discomfort of the parturient during labor and delivery is common practice. We have in our armamentarium a choice of paracervical, paravertebral, epidural or caudal block to provide analgesia during labor, and we may utilize pudendal, subarachnoid (saddle), epidural, caudal or field block to provide perineal anesthesia for delivery. In the last decade there have been major advances in our understanding of the effects of local anesthetics on both fetuses and neonates. For example, we now realize that local anesthetics may readily cross the placenta and produce undesirable responses in a fetus. Second, more sophisticated methods are available to study the subacute effects of local anesthetics on neonatal neurobehavioral function.

The initial interest in fetal and neonatal blood levels of local anesthetics was generated by studies documenting the occurrence of fetal bradycardia following paracervical block and a possible correlation of this event with blood levels of local anesthetics. Subsequent evidence suggests that the mechanism of fetal bradycardia associated with this procedure may be hypoxia caused by local anesthetic-induced uterine artery vasoconstriction. Consequently, paracervical block is losing popularity, and most recent investigations are concerned with epidural and caudal anesthesia. With these latter blocks, significant amounts of local anesthetic can be absorbed by the engorged epidural venous plexus of pregnancy. Similarly, pudendal block and local infiltration of the perineum may also result in significant uptake of local anesthetic into the maternal bloodstream due to the vascularity of the area. Moderate caution should be applied to these techniques.

When selecting an amide-type of local anesthetic agent requiring slow hepatic degradation, consideration should be given to those properties of the drug that allow for transfer from the maternal circulation across the placental membrane to the fetus. In addition, attention should be paid to the disposition and metabolism by the fetus and neonate because this determines ultimate toxicity. Substances that are highly bound to plasma proteins have less free drug available for transfer. Highly ionized or charged compounds do not cross membranes easily. Drugs which are highly lipid-soluble do cross membranes easily, but this same quality may allow for deposition in epidural adipose tissue, thereby decreasing the maternal level. In addition, because of rapid penetration into fetal fat compartments, the fetal blood level is also decreased.

The duration of action is important because the longer-acting agents tend to decrease the total quantity of drug required. Lidocaine and mepivacaine are considered intermediate in action with a duration of 45 to 75 minutes, while the long-acting bupivacaine (Marcaine) has a usual duration of 1½ to 2 hours. The short-acting ester 2-chloroprocaine (Nesacaine) has an average duration of 45 minutes. An additional advantage is its rapid rate of destruction by plasma cholinesterase and a half-life of 21 seconds. Little if any will be available for placental transfer. The